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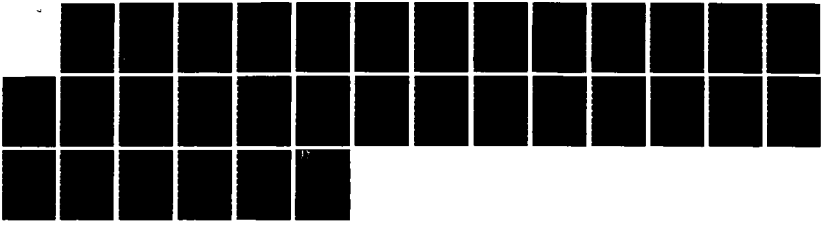

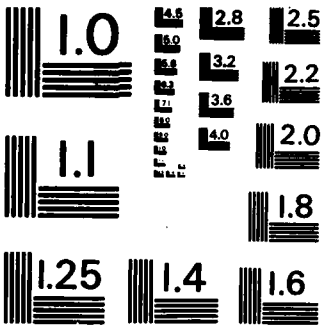

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During exercise in the heat, sweat output often exceeds water intake resulting in hypohydration which is defined as a body fluid deficit. This fluid deficit is comprised of water loss from both the intracellular and extracellular fluid compartments. Hypohydration during exercise causes a greater heat storage and reduces endurance in comparison to euhydration levels. The greater heat storage is attributed to a decreased sweating rate as well as a decreased cutaneous blood flow. These response decrements have been related to both plasma hyperosmolality and a plasma hypovolemia. Subject gender, acclimation

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state and aerobic fitness do not alter the hypohydration response. Hyper-hydration, or body fluid excess, does not appear to provide a clear advantage during exercise-heat stress, but may delay the development of hypohydration.

INFLUENCE OF HYDRATION LEVEL AND BODY FLUIDS ON EXERCISE PERFORMANCE IN THE HEAT

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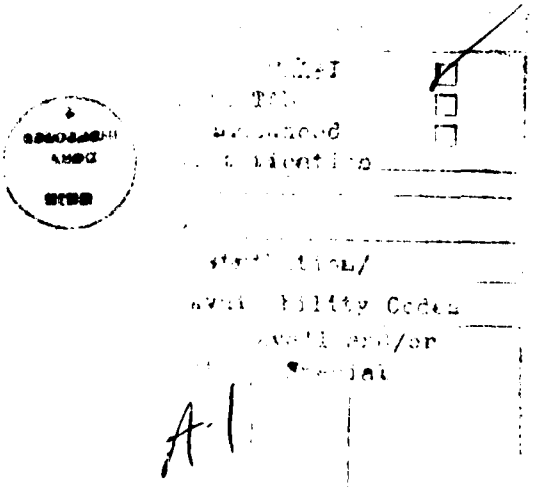
ABSTRACT

During exercise in the heat, sweat output often exceeds water intake resulting in hypohydration which is defined as a body fluid deficit. This fluid deficit is comprised of water loss from both the intracellular and extracellular fluid compartments. Hypohydration during exercise causes a greater heat storage and reduces endurance in comparison to euhydration levels. The greater heat storage is attributed to a decreased sweating rate as well as a decreased cutaneous blood flow. These response decrements have been related to both plasma hyperosmolality and a plasma hypovolemia. Subject gender, acclimation state and aerobic fitness do not alter the hypohydration response. Hyperhydration, or body fluid excess, does not appear to provide a clear advantage during exercise-heat stress, but may delay the development of hypohydration.

Key words: dehydration, heat stress, hormones, hyperhydration, hypohydration, osmolality, peripheral blood flow, plasma volume, sweating rate, temperature regulation, water requirements



During muscular exercise in the heat, thermoregulatory responses have been shown to be influenced primarily by aerobic fitness (1), acclimation state (2), and hydration level (3). Aerobically fit individuals who are heat acclimated and fully hydrated will have less body heat storage and optimal performance during exercise-heat stress. Hydration level is particularly important during exercise performance in the heat, since body fluid deficit will neutralize the thermoregulatory advantages conferred by aerobic fitness (4) and heat acclimation (5,6). Greenleaf (7) notes three body fluid levels: euhydration, hypohydration and hyperhydration. Euhydration refers to "normal" body fluid content; whereas, hypohydration and hyperhydration refer to body fluid deficit and excess, respectively (7). Greenleaf (7) defines the more common term "dehydration" as the dynamic loss of body fluids or the transition from euhydration to hypohydration.

BODY FLUID LOSS: In hot environments, body fluid is primarily lost via eccrine sweat gland secretion, which enables evaporative cooling of the body (8,9). Urinary fluid loss is decreased during heat stress relative to moderate environmental conditions (10,11). In addition, both exercise and hypohydration have been reported to decrease urine output below control levels (12). The volume of body fluid secreted as sweat can vary greatly. For a given individual, sweating rate is dependent upon environmental conditions (ambient temperature, dew point, radiant load, air velocity), clothing (insulation, moisture permeability) and physical activity level (10,13). Adolph and associates (10) reported that in a desert environment, soldiers performing normal daily activities had sweating rates ranging from 160 to 600 $g \cdot m^{-2} \cdot h^{-1}$. The amount of body fluid lost by sweat can be excessive, and sweating rates approaching 1000 $g \cdot m^{-2} \cdot h^{-1}$ are frequently reported (10,13,14). Table 1 provides an example of water

requirements for a 70 kg man working in a hot-dry environment (15). Dehydration will occur if the volume of fluid ingested is less than sweat output. However, the simple ingestion of fluids may not insure maintenance of euhydration since both exercise and heat stress result in large reductions in splanchnic blood flow (16), and thus reduce absorption rate.

TABLE 1 ABOUT HERE

Water intake may be equal to fluid loss over a 24-hour period in the heat (10). Within the 24-hour period in the heat, however, marked body water deficits may occur (10,11). Adolph and associates (10) reported that in the desert, euhydration was only re-established during the noon and evening meals. Apparently, food increases the palatability of water and assures more complete rehydration. Therefore, thirst may not provide a good index of body water requirements. Several studies (10,11,17,18) have reported that ad libitum water intake results in incomplete fluid replacement or "voluntary" dehydration during exercise in the heat. Recently, Hubbard et al. (19) examined the influence of water temperature and flavoring on "voluntary" dehydration during exercise-heat stress, and reported that water cooling and flavoring collectively increased water intake by 120%.

An individual's state of heat acclimation may also influence the level of "voluntary" dehydration incurred during exercise in the heat (17,20). Figure 1 presents data redrawn from Eichna et al. (17) on the "voluntary" dehydration (water deficit) incurred during exercise in a cool environment and during six consecutive days in a hot-dry (49^{o}C, 25% rh) environment. It can be noted that the water deficit was much greater during the initial exercise-heat exposures. In agreement with these findings, Greenleaf et al. (20) reported that water deficit

during exercise-heat stress was reduced by approximately 30% after the initial four days of heat acclimation. Therefore, heat acclimation may improve the relationship between thirst and body water needs (17, 20).

FIGURE 1 ABOUT HERE

As previously noted, sweat output is the primary avenue of body fluid loss during exercise in the heat. Sweat loss will result in a reduction of total body water if an adequate amount of fluid is not replaced. Total body water constitutes approximately 60% of an average adult's body weight (21), so a 70 kg individual would have a total body water of about 42 liters. Therefore, a fluid loss equal to 5% of body weight would constitute 8% of total body water for this individual. Total body water is divided into an intracellular and extracellular fluid compartment. The intracellular compartment is the sum of all fluids within cells, and the extracellular compartment includes all fluids outside of the cells. The intracellular fluid compartment contains 67% (28 liters) of total body water; extracellular fluid is distributed between the interstitium (26% or 11 liters) and plasma (7% or 3 liters). The fluid compartments are separated by water-permeable cell membranes which allow fluid exchange between compartments. Such exchange between compartments depends upon the gradients of colloid, tissue and hydrostatic pressures (21). As a consequence of this free fluid exchange, hypohydration should cause some water loss from each compartment.

The question arises as to how fluid loss is partitioned between these compartments during hypohydration. Costill et al. (22) have calculated the contribution of intracellular, interstitial and plasma fluid to the total body water loss in resting subjects at three hypohydration levels. Subjects were dehydrated

by 2%, 4% and 6% of total body weight using a combination of exercise and heat. Intracellular fluid accounted for 30% of the water loss at the 2% hypohydration level, and for 50% of the water loss at both the 4% and 6% hypohydration levels. Interstitial fluid accounted for 60% of water loss at the 2% hypohydration level, and for 40% of water loss at both the 4% and 6% hypohydration levels. Plasma fluid represented 10% of fluid loss at all three hypohydration levels. Therefore, at rest during low levels of hypohydration, water loss primarily occurred from the extracellular fluid compartment; however, at increasing hypohydration levels water loss was equally distributed between intra- and extracellular compartments.

Muscular exercise alters the colloid, tissue and hydrostatic pressure gradients between compartments resulting in additional fluid shifts. In particular, plasma volume shifts during exercise in the heat have been well studied (23-29). Hypohydration has been reported to either have no effect upon (24,26, 27), or to alter (27,28,29) the magnitude of vascular fluid shifts during exercise in the heat. Some of this inconsistency may be due to the varied protocols used to achieve fluid deficits. For example, several investigators elicited hypohydration with diuretics that caused iso-osmotic hypovolemia (24,29) as opposed to the hyperosmotic hypovolemia characteristic of hypohydration from sweating. Thus, vascular fluid shifts produced by diuretic-induced hypohydration may not be indicative of that which occurs during exercise in the heat. In addition, other investigators used a marginal hypohydration level (26) or did not report the hydration level (27). Recently, our laboratory (28) examined the effects of hypohydration (5%) on plasma volume shifts during exercise in the heat. For heat-acclimated subjects, we observed a hemoconcentration when hypohydrated and a hemodilution when euhydrated during light intensity exercise. Our study also demonstrated that when male and

female subjects are matched for maximal aerobic power, gender does not alter the vascular fluid shifts when either euhydrated or hypohydrated (28).

Eccrine sweat is ordinarily hypotonic relative to the plasma (30). Therefore, the plasma will become hyperosmotic when hypohydration is primarily mediated by sweat output (6,31). Plasma osmolality has been reported to increase from about 283 mosmol $\cdot kg^{-1}$ when euhydrated to levels approaching 300 mosmol $\cdot kg^{-1}$ when hypohydrated by exercise-heat stress (28,31). Sodium and potassium ions are primarily responsible for the elevated plasma osmolality during hypohydration (31). Several investigators have suggested that plasma hyperosmolality (32,33) as well as decreased plasma volume (29,34) contribute to the less efficient thermoregulatory responses when hypohydrated during exercise in the heat. However, such fluctuations in osmolality and plasma volume are the triggering mechanism for adaptive responses to conserve body fluids.

HORMONAL RESPONSES: Ordinarily, a decreased plasma volume and simultaneous elevation in plasma osmolality stimulate hypothalamic osmoreceptors initiating neural responses which increase vasopressin secretion and stimulate the thirst sensation (21,35). Consequently, vasopressin increases water reabsorption by the renal distal tubules, thus conserving vascular fluid and attenuating the dehydration process. Animal studies indicate that the renin-angiotensin system also stimulates the release of vasopressin and aldosterone, and increases the sensation of thirst. For example, Severs et al. (36) reported that the central administration of angiotensin II to rats was as effective in stimulating drinking behavior as cellular dehydration or hypovolemia. Mann et al. (37) demonstrated that a circulating angiotensin II concentration of approximately 200 fmol $\cdot ml^{-1}$ represents the dipsogenic threshold for rats. Thus, neural and endocrinological mechanisms for stimulated water conservation and consumption are operative under a variety of hypohydration influences.

There are many reports documenting that circulating concentrations of the fluid-electrolyte regulatory hormones are elevated in man during acute heat exposure or even during exercise in a cool environment. For example, elevations in circulating levels of aldosterone, renin activity, cortisol and vasopressin during sedentary exposure to several different thermal stresses have been observed (38,39,40). Likewise in exercising humans without heat stress, several investigators have documented increments in the same group of hormones (41,42,43). These hormonal responses are modulated by heat acclimation (44), saline loading (45), potassium supplements (46) and physical training (42).

Our laboratory examined the effects of hypohydration and heat acclimation on circulating aldosterone and renin activity during exercise in several environments (47). When at rest in a moderate environment, hypohydration significantly elevated circulating aldosterone and plasma renin activity. We also observed that plasma renin activity was significantly elevated during exercise-heat stress and that these increments were markedly accentuated by hypohydration and attenuated by heat acclimation. Further, the increased aldosterone levels observed during exercise in the heat were even greater during hypohydration. Heat acclimation did not influence the aldosterone hormonal responses (47). Conversely, in experiments in which plasma volume was expanded by hyperoncotic albumin administration, we reported reduced responses in aldosterone and angiotensin I levels at several sampling intervals during exercise in the heat (48).

It is apparent that humans manifest a variety of endocrinological adaptations designed to stimulate fluid consumption and reduce fluid and electrolyte loss during thermal and exercise stress. Contributing to the prevention of hypohydration and the maintenance of fluid homeostasis are hormonally mediated responses including increases in the sensitivity of the thirst

mechanism and elevated antidiuretic and antinatriuretic activity. Less studied have been the effects of hormones on gastric emptying, intestinal absorption and the partitioning of water loss among the body fluid compartments during several hypohydration levels.

PHYSIOLOGICAL RESPONSES: Adolph and associat[illegible]10) in their book entitled Physiology of Man in the Desert describe the symp[illegible] associated with hypohydration in the desert. They state that these symptoms occur throughout a broad range of water deficits (as represented by percent decrease of body weight), and that there is considerable intersubject variability for these symptoms. Thirst is reported to occur after a 2% water deficit, but does not increase in intensity with greater hypohydration levels. A 4% to 6% water deficit is associated with anorexia, impatience and headache; whereas, a 6% to 10% water deficit is associated with vertigo, dyspnea, cyanosis and spasticity. An individual who has incurred more than a 12% water deficit will be unable to swallow and will need assistance with rehydration. Adolph and associates (10) estimated that depending upon environmental conditions and exposure time, the lethal hypohydration level is between 15% to 25% water deficit.

Table 2 presents a summary of investigations examining the thermoregulatory effects of hypohydration for resting subjects in the heat (31,49-52). These investigations were selected to represent a continuum from a marginal (1%) to the largest (6%) level of fluid deficit reported for these conditions. Caution should be employed when comparing the results of different investigations because of differences in subject populations (age, fitness, acclimation state, gender), environmental conditions (ambient temperatures, dew point) and duration of water deficit. The equal signs in the table denote no significant difference, while the arrows represent the direction of the significant

difference between euhydration and hypohydration. A marginal water deficit (1-2%) does not appear to alter resting values for heart rate (HR), core temperature (T_c), sweating rate ($\dot{M}_{sw}$) or peripheral blood flow (PBF). However, hypohydration levels of 3% to 6% resulted in significantly elevated heart rate, core temperature, and reduced sweating rate and peripheral blood flows than during euhydration experiments. In addition, a trend for greater increments in core temperature appeared to occur for the 6% than 3% water deficit.

TABLE 2 ABOUT HERE

Gender (6), acclimation state (6) and aerobic fitness (4) do not alter the elevated thermoregulatory responses when hypohydrated during exercise in the heat. Table 3 presents a summary of investigations examining the thermoregulatory effects of hypohydration for subjects performing light intensity exercise in the heat (3,6,15,24,34,49,53,54). Again, these investigations represent a continuum from marginal (2%) to the largest (7%) water deficits reported for these conditions. As with Table 2, it is tenuous to make direct comparisons among the studies. Unlike rest (Table 2), a 2% water deficit during exercise significantly reduced sweating rate by 3%, and elevated both heart rate by 10 bpm and core temperature by 0.5°C in comparison to euhydration levels. It can be noted that considerable inter-investigation variability exists for the magnitude of elevated heart rate when hypohydrated. For example, a 3% water deficit elevated heart rates by a range of 8-40 bpm. This variability is probably in part accounted for by differences in subject populations, exercise intensity and duration, and environmental conditions. In contrast, little variability was

found for the increment in core temperature when hypohydrated; a 2% and 7% water deficit elevated core temperatures by 0.5°C and 0.7°C above euhydration levels, respectively.

TABLE 3 ABOUT HERE

It is generally assumed that with increasing hypohydration level there is a gradation of elevated core temperature response. It is possible, however, that the elevated core temperature response could represent a "threshold" or "all or none" effect. We are not familiar with research that thoroughly examines the effects of different hypohydration levels on the magnitude of elevated core temperature responses during exercise in the heat. Strydom and Holdsworth (3) examined core temperature elevations for two hypohydration levels (low and high) employing independent observations for each exercise-heat test; however, they only had 2 subjects and used a broad range of water deficits for the low (3-5%) and high (5-8%) hypohydration tests. They found significantly higher core temperatures during the high than low hypohydration tests. Other studies reporting values for a gradation of elevated core temperatures with increasing water deficits have interpolated from one hypohydration level (55) and/or employed prolonged exercise-heat exposure to elicit a progressive dehydration (10,56). Therefore, research is needed on the effects of different hypohydration levels on thermoregulatory responses during exercise in the heat.

The relative contribution of evaporative (E_{sk}), radiative and convective heat exchange during exercise depends upon the specific environmental conditions. When ambient temperature approaches body temperature, E_{sk} provides the primary mechanism to dissipate metabolic heat during exercise.

Table 3 indicates that hypohydration is associated with reduced (3,6,53,54) or unchanged (15,24,49) sweating rates during exercise-heat stress. It is important to note that some investigators reported that there was no change in sweating rate, despite significantly elevated core temperature (24,49). Therefore, during hypohydration sweating rate would be lower for a given core temperature, and the potential for heat dissipation via E_{sk} would be reduced (8). These findings are consistent with recent data which indicate that hypovolemia caused reduced slope ($\Delta \dot{M}_{sw} / \Delta T_c$) of sweating rate responses during exercise (29). Therefore, a reduced slope for the sweating rate response would result in an elevated core temperature during exercise-heat stress.

The exact physiological mechanisms mediating the reduced sweating rate response when hypohydrated are not clearly defined. However, the singular and combined effects of plasma hyperosmolality (54,57,58) and hypovolemia (29,50) have been suggested. Senay (54) has reported a significant inverse relationship between plasma osmolality and the sweating rate when hypohydrated. Harrison et al. (59) have presented data indicating that plasma hyperosmolality will elevate core temperature responses during exercise-heat stress, despite the maintenance of euhydration. Hyperosmolality may have a direct central nervous system effect at the hypothalamic thermoregulatory centers (33), or a peripheral effect at the eccrine sweat gland (57). Fortney et al. (29) have provided strong evidence that an iso-osmotic hypovolemia caused a reduced sweating rate and elevated core temperature response during exercise in the heat. These investigators theorized that hypovolemia may have altered the activity of atrial baroreceptors which have afferent input to the hypothalamus. Therefore, a reduced atrial filling pressure might modify neural information to the hypothalamic thermoregulatory centers controlling sweating rate (29).

The influence of hypohydration on cardiovascular responses to exercise has been investigated (34,60,61). During submaximal exercise in a neutral environment, hypohydration (5%) elicited increased heart rate and reduced stroke volume with no change in cardiac output relative to euhydration levels (60). Apparently, during hypohydration, a decreased blood volume reduces the end diastolic ventricular volume and stroke volume, requiring a compensatory increase of heart rate to maintain cardiac output. During submaximal exercise with moderate (34) or severe (61) thermal strain, hypohydration (3 to 4%) elicited increased heart rate, reduced stroke volume and reduced cardiac output relative to euhydration levels. The combination of exercise and heat strain results in competition between central and peripheral circulation for a limited blood volume (16,61). As body temperature increases during exercise, cutaneous vasodilation occurs thus decreasing venous resistance and pressure. As a result of decreased blood volume and blood displacement to cutaneous vascular beds, venous return and thus cardiac output will be decreased below euhydration levels (34,61). Nadel et al. (34) reported that these conditions also reduce cutaneous blood flow for a given core temperature and thus the potential for radiative and convective heat exchange.

Table 4 presents the effects of hypohydration on maximal aerobic power and physical work capacity (PWC). In the absence of heat stress, a relatively large water deficit (6 to 7%) has a minimal effect on maximal aerobic power (5,62), but reduces physical work capacity (62). In a hot environment, Craig and Cummings (63) demonstrated that small (2%) to moderate (4%) water deficits significantly reduce maximal aerobic power and physical work capacity. In addition, these decrements increased with the magnitude of the water deficit. Consistent with these findings, hypohydration (4%) combined with hyperthermia in a moderate environment, significantly reduced maximal aerobic power by 6%

and exercise time by 12% from euhydration levels (64). These investigations clearly demonstrate that maximal exercise performance is reduced when hypohydration is combined with thermal strain. The physiological mechanism responsible for reduced aerobic power is probably an inability to maximally increase cardiac output (and hence oxygen delivery).

TABLE 4 ABOUT HERE

HYPERHYDRATION: If hypohydration reduces performance during exercise-heat stress, can excess body fluids improve performance beyond the levels achieved when euhydrated? Moroff and Bass (53) examined the influence of excessive fluid ingestion on thermoregulatory responses to exercise in the heat. They reported that hyperhydration significantly reduced core temperatures while elevating sweating rates above control levels. During the control experiments, however, their subjects were slightly (> 1%) hypohydrated. Therefore, these results may have demonstrated the effects of hypohydration rather than hyperhydration. More recently, Greenleaf and Castle (55) reported that excessive fluid ingestion did not alter core temperature or sweating rate values from control levels during exercise in the heat.

If hyperhydration did improve performance during exercise-heat stress, these improvements would most likely be mediated by hypervolemia or expanded blood volume. In fact, some of the thermoregulatory advantages gained through heat acclimation have been associated with an expanded plasma volume (65). Two recent studies on the effects of artificially expanded plasma volume have reported no differences in core temperature (34,66), sweating rate (66), cardiac output (34), arterial blood pressure (66), or peripheral blood flow (34) in

comparison to normo-volemic control levels during exercise-heat stress. Both studies found that plasma volume expansion lowered heart rate responses during exercise in the heat (34,66). In contrast, Fortney et al. (29) reported that an artificially expanded blood volume significantly lowered core temperature below control levels during exercise. This was observed despite no difference in sweating rate in a 30°C environment. These studies indicate that the thermoregulatory advantages of hyperhydration are quite small and may be affected by the protocol inducing hypervolemia.

SUMMARY: A relevant topic to the 1984 Summer Olympics is the influence of hydration and body fluids on exercise performance. Of particular interest are distance running events requiring prolonged performance at high metabolic rates in hot environments. The potential for dehydration is great, as marathon runners have been reported to incur fluid deficits in the range of 4 to 7% of body weight (67,68). As discussed, hypohydration will increase thermal strain above the level encountered with euhydration. Continued athletic performance with incomplete rehydration will initiate a cycle of elevated core temperature causing greater thermal drive for sweating and further dehydration (15). This cycle could lead to heat exhaustion or even heat stroke (69). It has been recommended that runners ingest fluid prior to competition to insure euhydration, and frequently ingest fluids during competition to help prevent heat injuries during long distance running (69). Knowledge of the physiological actions of hypohydration should help physicians to prevent such heat injuries.

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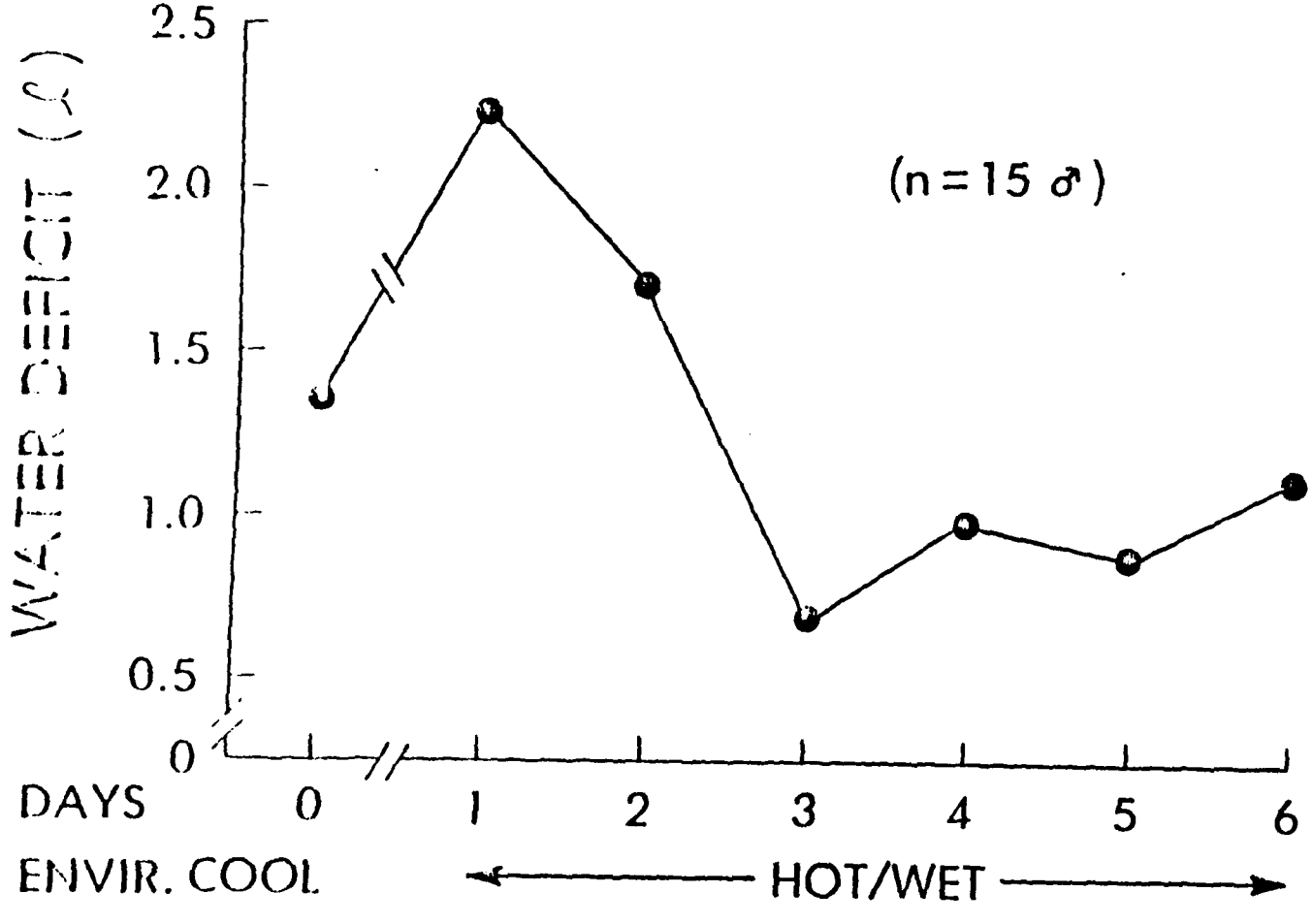
WATER REQUIREMENTS OF MEN LIVING AND WORKING IN THE HEAT (~90°F/30%rh)

A. SLEEPING	8 HOURS	=	0.5 ℓ
B. SEMI ACTIVE	8 HOURS	=	1.0 ℓ
C. MARCHING 6.8 kph WITH PACKS	5 HOURS	=	4.6 ℓ
D. PREPARATION ACTIVITIES	3 HOURS	=	0.9 ℓ
TOTAL =	24 HOURS	=	7.0 ℓ

FROM THE DATA OF STRYDOM et al 1966.

DIFFERENCE BETWEEN AD LIB WATER INTAKE AND SWEAT LOSS DURING WORK IN HUMID HEAT



REDRAWN FROM THE DATA OF EICHNA et al 1945

EFFECTS OF HYPOHYDRATION DURING REST IN THE HEAT

STUDY	YEAR	% Δ WT	HR	T_C	$\dot{M}_{sw}$	PBF
SWAMY et al	1981	-1%	=	=	=	
SENAY & CHRISTENSEN	1965	-2%			=	=
HERTZMAN & FERGUSON	1960	-3%	↑ 8 bpm	↑ 0.6°C	=	
MYRE & ROBINSON	1977	-3%		=	=	
SWAMY et al	1981	-3%	↑ 17 bpm	↑ 0.4°C	↓ 22%	
HORSTMAN & HORVATH	1972	-4%		↑ 1.0°C	↓ 21%	↓
HERTZMAN & FERGUSON	1960	-6%	↑ 17 bpm	↑ 1.2°C	=	
SENAY & CHRISTENSEN	1965	-6%			=	=

EFFECTS OF HYPOHYDRATION DURING EXERCISE IN THE HEAT

STUDY	YEAR	% Δ WT	HR	T_C	$\dot{M}_{sw}$	PBF
MOROFF & BASS	1965	-2%	↑ 10 bpm	↑ 0.5°C	↓ 3%	
CLAREMONT et al	1976	-3%	↑ 20 bpm	↑ 0.6°C	=	↓
NADEL et al	1980	-3%	↑ 8 bpm	↑ 0.4°C		↓
STRYDOM et al	1966	-3%		=	=	
SWAMY et al	1981	-3%	↑ 40 bpm	=	=	
SENAY	1968	-4%			↓ 17%	
STRYDOM et al	1968	-4%	↑ 19 bpm	↑ 0.3°C	↓ 33%	
SAWKA et al	1983	-5%	↑ 13 bpm	↑ 0.5°C	↓ 13%	
		-5%	↑ 16 bpm	↑ 0.8°C	↓ 13%	
SWAMY et al	1981	-6%	↑ 60 bpm	↑ 0.8°C	=	
STRYDOM et al	1968	-7%	↑ 29 bpm	↑ 0.7°C	↓ 33%	

EFFECTS OF HYPOHYDRATION ON MAXIMAL EXERCISE PERFORMANCE

STUDY	YEAR	TEST ENVIRON.	% Δ WT	$\dot{V}O_2$ max	PWC
CRAIG & CUMMINGS	1966	HOT	-2%	↓ 10%	↓ 22%
			-4%	↓ 27%	↓ 48%
BUSKIRK et al	1958	NEUT.	-6%	↓ ?	
SALTIN	1964	NEUT.	-7%	=	↓ 20%

HUMAN RESEARCH

Human subjects participated in these studies after giving their free and informed voluntary consent. Investigators adhered to AR 70-25 and USAMRDC Regulation 70-25 on Use of Volunteers in Research.

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